Meigs' Syndrome: Report of a Typical Case

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In 1937 Meigs and Cass[2] reported seven cases of fibroma of the ovary associated with ascites and hydrothorax. They believed theirs to be the largest series ever reported. In 1954, Meigs[4] redefined the syndrome as being limited to cases with: (1) Either fibroma of the ovary, thecoma, granulosa cell tumor or Brenner tumor; (2) ascites; (3) hydrothorax; and (4) cure after the removal of the tumor. In the same year Meigs[5] reviewed reports of 84 cases of the syndrome as thus defined, the collection embracing all the English language literature and some of the cases reported in other languages. Since 1954, reported cases have been few.

One must keep in mind that almost any sort of ovarian lesion may produce ascites and hydrothorax. It has been reported that fibroma and fibroma-like tumors of the ovary are by far the most common cause of this syndrome, but among ovarian tumors in general they are of relatively low incidence. This seems to be indicative that there is something special about the ability of fibroma and fibroma-like tumors to produce ascites and hydrothorax. More important is the realization that these benign tumors can mimic a malignant lesion, often producing in the patient what appears to be a terminal state due to widespread metastatic process.

The cause of the ascites has remained a controversial subject. Wallingford[6] proposed a possible hormonal origin of Meigs' syndrome. However, the cause of the abdominal fluid is probably best explained by the older concept that the edema within the tumor is the source of the ascites. Numerous investigators have reported that most fibromas associated with Meigs' syndrome show microscopic evidence of edema; other observers, placing a fibroma in a dry receptacle, noted a reduction in the weight of the specimen due to a loss of fluid. Whether the edema is due to pressure of the firm tumor on the efferent blood and lymphatic vessels in the pedicle or hilum of the ovary or is brought about in some other way is not known.

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Submitted June 29, 1961.

The source of the fluid in the chest is less speculative. Meigs and others have shown with the use of electrophoretic protein determination that the chest and abdominal fluids are the same. Meigs, Armstrong and Hamilton,[3] and Lawson,[1] by means of India ink injection have demonstrated that the passage of fluid is from the abdomen to the chest and not the reverse. Cron, in discussing a paper by Meigs[5] mentioned a patient with an ovarian liposarcoma and associated extensive ascites and bilateral pleural effusion. Radioactive colloidal gold was injected intraperitoneally and recovered from the pleural cavities as early as two hours after administration.

The signs and symptoms of Meigs' syndrome are generally typical. Dyspnea, chest pain, evidence of fluid in the chest on physical examination, abdominal swelling and pain, and edema of the legs are all findings that should make the observer consider this syndrome. Neck vein distention, mediastinal shift, hepatomegaly, incontinence and frequency of urination, abnormal uterine bleeding of all types, and uterine prolapse have been reported as associated findings.

Confirmation of the syndrome may be aided by diagnostic studies demonstrating the tumor, ascites and hydrothorax. Cytologic examination of the abdominal and chest fluid help to rule out a malignant process.

Treatment of the patient consists of the removal of the tumor.

REPORT OF A CASE

A 73-year-old white widow was admitted, on referral, to the clinic service of the Queen of Angels Hospital on May 23, 1960, because of exertional dyspnea, weakness, increase in abdominal size and "something bulging from the vagina." The patient was belligerent but well oriented. She said she had never had heart or lung disease. Orthopnea when lying on the left side, and exertional dyspnea had been present for four weeks. She said her appetite had remained good but that her abdomen had increased in size over the five previous weeks. She did not have nausea or abdominal pain, and she did not know whether or not she had lost weight. Bowel movements had occurred daily until approximately two weeks before admission; from that time evacuation would occur on every second or third day.

The patient said the stool was firm but she had not noted the color. Nocturia occurred twice nightly; there was no dysuria, urgency, frequency or stress incontinence. The patient was gravida 1, para 1. She had last menstruated 25 years previously. Before the present admittance to hospital she had noted a slight pinkish vaginal staining and a bulging from the vagina, both of five weeks' duration, the bulging making walking difficult. She said she had never had a surgical operation and was not taking medication.

Upon physical examination the patient did not seem uncomfortable. Her height was 5 feet 4½ inches and her weight 89 pounds. The skin was warm and dry with poor turgor. The chest was hyperresonant to percussion on the left, and dull to percussion on the right. Breath sounds on the right were distant. The pulse rate was 80 and regular and the blood pressure 130/80 mm. of mercury. There was no evidence of increase in cardiac size. Heart sounds were distant and not audible in the aortic and pulmonic areas. The abdomen was rotund, soft, and without tenderness. Suggestion of a fluid wave was present. There was a firm, nontender, irregular suprapubic mass extending to the umbilicus and filling the right lower quadrant of the abdomen. A ballotable mass that occupied part of the right upper quadrant was palpable above the umbilicus. The liver, kidneys and spleen were not palpable. Upon pelvic examination, complete procidentia with ulceration of the vaginal mucosa was noted. The cervix was clean, without evidence of bleeding. The uterine cavity was sounded to 3½ inches. Further evaluation of the intrapelvic contents was impossible, as the pressure and fullness behind the protruding structures prevented reduction of the prolapse. Soft, tan stool was noted on rectal examination. A trace of pretibial edema was present. No abnormalities were observed on neurologic examination.

The clinical impression at this time was probable ovarian carcinoma with ascites and hydrothorax secondary to metastasis.

Hemoglobin content was 13.3 gm. per 100 cc. of blood. Leukocytes numbered 8,800 per cu. mm., with the cell differential within normal limits. Erythrocytes and platelets appeared normal. Routine examination of a voided specimen of urine was reported as showing specific gravity of 1.010, acid reaction, negative reaction for sugar and ketone bodies. There was a trace of albumin, 1 to 2 leukocytes and 10 to 15 erythrocytes per high power field. Blood urea nitrogen was 10 mg. per 100 cc., albumin 2.9 gm. and globulin 3.7 gm. per 100 cc. Blood electrolytes were well within normal limits. An electrocardiogram was compatible with anterior lateral myocardial ischemia which could represent left ventricular hypertrophy. Five days before admission to hospital, an abdominal paracentesis had been done at the patient's home and 60 cc. of straw-colored fluid was withdrawn. A cytologic examination of a prepared specimen of the fluid was negative for malignant disease.

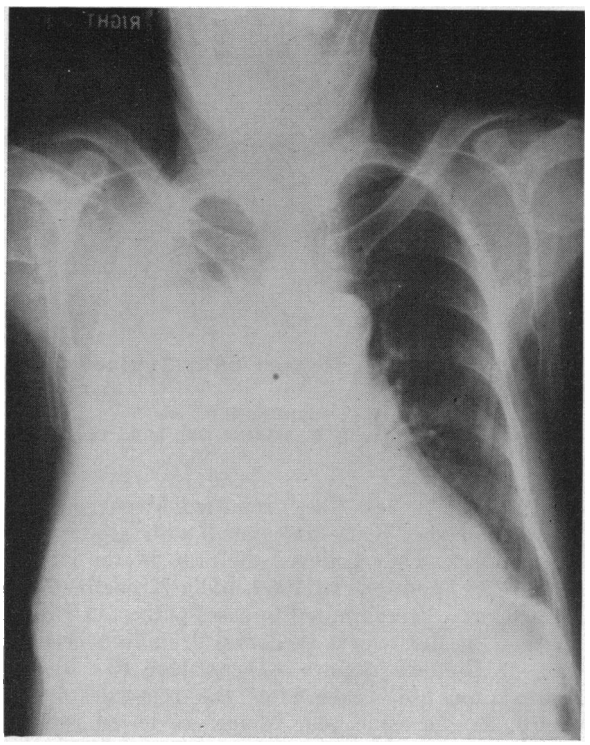

Figure 1.—Roentgenogram showing opacification of the right hemithorax by pleural fluid.

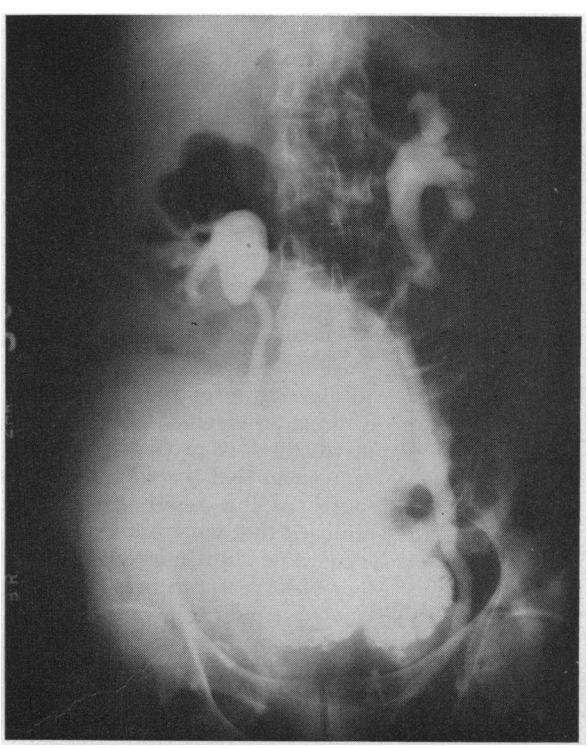

Figure 2.—Roentgenogram demonstrating bilateral hydronephrosis and hydroureter. Calcified leiomyomata overlie the pelvis. The ovarian fibroma displaces the intestinal loops to the left.

On May 24, roentgenograms of the chest (Figure 1) confirmed the presence of extensive pleural fluid on the right which opacified almost the entire hemithorax except for a small area in the apical region. As there was neither displacement of the mediastinal structures nor engorgement of the central lung vessels, the fluid was believed not to be from the circulatory system. Thoracentesis was carried out and 1,900 cc. of pale yellow, slightly turbid fluid was removed, which made breathing easier. Cytologic examination of the fluid revealed no evidence of malignant disease.

On May 25, roentgen examination of the chest showed less pleural fluid than on the previous day. The heart did not appear to be enlarged. The left lung field was clear and the bones of the thorax intact. An excretory urogram (Figure 2) showed good infusion of the contrast media on both sides. There was bilateral hydronephrosis and hydroureter. The ureters appeared to enter the bladder somewhat below the level of the symphysis. Renal drainage was quite poor and a half hour after the first urogram the renal pelves were still considerably distended. Overlying the pelvic inlet were two large calcific masses that were believed to be calcified uterine leiomyomata. An additional mass appeared to extend above the pelvic inlet into the abdominal cavity and displace the intestinal loops to the left; it was believed to be a cyst, possibly of ovarian origin. No definite conclusion could be reached as to whether or not intra-abdominal fluid was present.

Two days later, arm-to-tongue circulation time with dehydrocholic acid (Decholin®) was 21 seconds. Venous pressure was 7.5 cm. of water. A consultant believed that the patient might be in early left ventricular failure although compensated. Digitalization was recommended and completed.

On May 30 an additional 1,400 cc. of fluid was removed from the right chest. Cytologic examination was negative for malignant cells.

On June 2, with the patient under general anesthesia, cytoscopy was carried out. Mild trigonitis, an intact mucosa, pronounced distortion of the bladder wall secondary to the intrapelvic tumor and decided prolapse of the uterus were noted. Urine was observed to appear at both ureteral orifices.

On June 8 an additional 800 cc. of fluid was removed from the right chest. At this time a diagnosis of Meigs' syndrome was considered plausible because of inability to prove a malignant lesion.

On June 9 with the patient under epidural anesthesia, the abdomen was opened through a subumbilical, right paramedian incision. 600 cc. of clear yellow intraperitoneal fluid was present. A left ovarian tumor (Figure 3) 17x16x12 cm. was removed.

For expediency, supracervical hysterectomy rather than a total hysterectomy was carried out, the excised uterine specimen measuring 12x8x9 cm. The right ovary and tube were removed. The ovary, 4 cm. in diameter, had a firm, pink, 8 mm. nodule bulging from its surface. The remaining abdominal viscera appeared normal. Wire retention sutures were used in closing the abdomen. Estimated blood loss was 200 cc. The patient tolerated the procedure well.

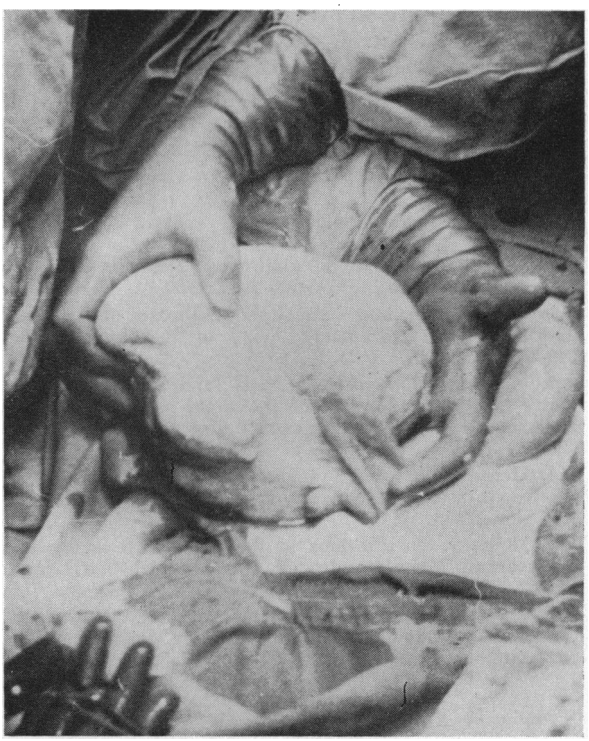

Figure 3.—Photograph at operation, showing the ovarian fibroma which measured 17x16x12 cm.

The pathological description was consistent with bilateral ovarian fibromas. Some edema was noted in one of the sections. A stain for fat was negative. The uterus contained partially calcified leiomyomata and hyperplastic endometrium of the Swiss cheese type.

The only postoperative complications were a single episode of auricular fibrillation and a urinary tract infection which was treated successfully with a sulfa drug.

X-ray studies on June 29 showed the right diaphragm slightly elevated and the right costophrenic angle somewhat obscured, probably by the presence of pleural fluid. Considerable improvement was evident. An intravenous pyelogram showed the kidneys functioning normally and regression of the previously described bilateral hydronephrosis. The ureters were well outlined and appeared to be of normal caliber.

Surprisingly, upon vaginal examination before the patient was discharged from hospital, fair support of the vaginal vault was noted despite the fact that at operation no effort was made to correct the prolapse.

On July 17 the patient was discharged to her home. In the course of the year following the operation, the patient's preoperative belligerence was

supplanted by friendliness and complete cooperation. She gained 25 pounds and felt well. The only problem was recurrence of vaginal prolapse shortly after she left the hospital. It was corrected with a No. 4 ring pessary, but she had a minor degree of stress incontinence with the pessary in place.

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REFERENCES

1. Lawson, J. G.: Meigs' syndrome, three cases, J. Obst. & Gynec., Brit. Emp., 57:595-604, Aug. 1950.

2. Meigs, J. V., and Cass, J. W.: Fibroma of the ovary with ascites and hydrothorax, Am. J. of Obst. & Gynec., 33: 249-267, Feb. 1937.

3. Meigs, J. V., Armstrong, S. H., and Hamilton, H. H.: Meigs' syndrome, fibroma of the ovary with fluid in the abdomen and chest, further contribution, Am. J. of Obst. & Gynec., 46:19-37, July 1943.

4. Meigs, J. V.: Pelvic tumors other than fibromas of the ovary with ascites and hydrothorax, Obst. & Gynec., 3:471-486, May 1954.

5. Meigs, J. V.: Fibroma of the ovary with ascites and hydrothorax, Meigs' syndrome, Am. J. of Obst. & Gynec., 67:962-987, April 1954.

6. Wallingford, A. J.: Ascites in gynecology with special reference to a possible hormonal origin of Meigs' syndrome, N. Y. State J. of Med., 55:221-227, Jan. 1955.

Ruptured Congenital Aortic Sinus Aneurysm

Report of a Case Successfully Corrected Following Aortographic Demonstration

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THE SUCCESS of operations on the heart with the surgical field exposed to view has much increased the obligation to make precise diagnosis of corrigible lesions. Aortocardiac fistula from rupture of congenital aneurysm of an aortic sinus (sinuses of Valsalva) has been successfully repaired in 13 reported cases since 1957.* A recent review[7] listed 59 cases reported up to 1958. Most of them were diagnosed postmortem.

We have found reports of aortographic demonstration of only five such cases. In the first proven case, reported by Brofman and Elder in 1957, retrograde aortography showed a fistula from the sinus of Valsalva to the right ventricle. We are reporting another case.

The patient was a girl 17 years old. She had developed well and had easily kept up with children of her age. In early childhood she had repeated mild respiratory infections. Cardiac murmurs had been present since birth, but no cyanosis nor any other sign or symptom of disease.

She appeared normal and healthy, with no edema, cyanosis or clubbing of fingers. All the classical signs of a wide pulse pressure were present—bobbing head, collapsing pulse, visible capillary pulsation, pistol shot audible over both femoral arteries. The chest bulged to the left of the sternum. A thrill was palpable from the second intercostal space to the cardiac apex, systolic in time but extending into diastole. The heart was large to percussion. The apical impulse was rolling and very vigorous with the point of maximal impulse out to the anterior axillary line in the fifth interspace. The first heart sound was louder in the aortic area than in the pulmonic, but the second aortic sound was barely audible. The second pulmonic sound was loud, not split. A harsh systolic murmur was heard all over the precordium—very loud at the pulmonic area, and was transmitted to the whole left chest and back. A murmur arising directly with the second sound occupied the whole of diastole. In the third left interspace it took on the quality of a machinery murmur —continuous and very loud. Systolic blood pressure (brachial) was 150; diastolic zero to 30 mm. of mercury.

Radiologically both ventricles were enlarged. The pulmonary segment stood out and the pulmonary vessels appeared engorged. The aortic knob was small, as were the ascending and descending aorta. Left atrial enlargement displaced the esophagus backward and produced a double contour at the right heart border (Figure 1). The electrocardiogram was consistent with right ventricular hypertrophy.

A cardiac catheter threaded into the right femoral vein passed through an interventricular septal defect and on into the ascending aorta. This happened on two occasions. The passage was not through a fistula from sinus to right ventricle, for the catheter was seen definitely within the left ventricle before passing into the aorta. The large left to right shunt was obvious. The high pressure gradient from the right ventricle to the pulmonary artery we thought was due to the very large volume of blood passing through the pulmonic valve. We thought likely also that there was a defect in an aortic leaflet with some regurgitation (see Table 1).

TABLE 1.—*Data from Cardiac Catheterization*

Source	O_2 Content (Vol. Per Cent)	Pressures (mm. Mercury) Systolic	Diastolic
Right pulmonary artery	14.0	.....	....
Main pulmonary artery..	14.4	21	11
High in right ventricle..	14.2	66	7
Low in right ventricle....	14.3	.....	....
Low in right atrium.....	10.3	.....	....
High in right atrium.....	10.2	.....	....
Superior vena cava.....	10.1	.....	....
Inferior vena cava.....	10.5	.....	....
Left venrticle	15.0	101	11
Aorta	14.9	101	65

	Liters per Minute
Pulmonary blood flow.....	26.0
Systemic blood flow.....	4.5
Left-to-right shunt	21.5

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Submitted July 21, 1961.

*Reference Nos. 1, 6, 7, 8, 9, 10, 12, 13.